WHAT IS CLAIMED IS:

1. A method of compiling a functional gene profile of a donor organism, comprising:

(a) introducing into an episomal non-transforming non-viral vector a mixture of a donor organism derived DNA or RNA sequences to construct an episomal non-transforming non-viral vector-based library, wherein the sequences are unidentified, wherein each member of the library comprises an insert from the mixture;

(b) introducing into a host said one or more members of the library;

(c) transiently expressing said unidentified nucleic acid in the host;

(d) determining one or more phenotypic or biochemical changes in the host;

(e) identifying an associated trait relating to said one or more phenotypic or biochemical changes;

(f) identifying the member that results in said one or more changes in the host;

(g) repeating steps (b) – (f) until at least one nucleic acid sequence associated with said trait is identified, whereby a functional gene profile of the host or of the donor organism is compiled.

2. A method of identifying the sequence of an antigen of a pathogen, which when expressed in a host confers immuno-protection on the host against the pathogen, comprising:

(a) introducing into an episomal non-transforming non-viral vector a mixture of the pathogen derived DNA or RNA sequences to construct an episomal non-transforming non-viral vector-based library, wherein each member of the library comprises an insert from the mixture;

(b) introducing the library into a group of hosts wherein each host contains one member;

(c) expressing each insert, capable of expression in the host, in the host in which the member resides;

(d) challenging each of the host with the pathogen;

(e) determining which host has immuno-protection against the pathogen; and

(f) determining the sequence of the insert in the host determined in step (e);

whereby the sequence of the antigen of the pathogen is identified.

3. A method of identifying the sequence of an antigen of a cancer cell, which when expressed in a host confers immuno-protection on the host against the cancer cell, comprising:

(a) introducing into an episomal non-transforming non-viral vector a mixture of the cancer cell derived DNA sequences to construct an episomal non-transforming non-viral vector-based library, wherein each member of the library comprises an insert from the mixture;

(b) introducing the library into a group of hosts wherein each host contains one member;

(c) expressing each insert, capable of expression in the host, in the host in which the member resides;

(d) challenging each of the host with the cancer cell;

(e) determining which host has immuno-protection against the cancer cell; and

(f) determining the sequence of the insert in the host determined in step (e);

whereby the sequence of the antigen of the cancer cell is identified.

4. The method according to Claim 1, wherein the episomal non-transforming non-viral vector comprises a replication-competent, transformation-negative vector comprising at least one papovavirus origin of replication, a first DNA sequence encoding a mutant form of papovavirus large antigen which contains a replication-competent binding site for the origin of replication and which is negative for binding to and to retinoblastoma tumor suppressor gene product due to a mutation in a codon in the p53 binding domain of the large T antigen and a mutation in a codon

in the RB binding domain of the large T antigen, the DNA sequence being operatively linked to a first promoter which is functional in the host cell, and a second DNA sequence encoding the foreign gene operatively linked to a second promoter which is function in the host.

5. The method according to Claim 4, wherein the papovavirus origin of replication is a BK virus origin of replication or a SV40 origin of replication.

6. The method according to Claim 5, wherein the mutant form of the large T antigen contains a replication competent binding site for both the BK virus origin of replication and the SV40 origin of replication.

7. The method according to Claim 4, wherein the replication-competent, transformation-negative vector further comprises a bacterial origin of replication.

8. The method according to Claim 4, wherein the first promoter is inducible.

9. The method according to Claim 4, wherein the first promoter is constitutive.

10. The method according to Claim 4, wherein the first promoter is under hormonal control.

11. The method according to Claim 1, wherein the episomal non-transforming non-viral vector comprises a DNA sequence encoding a mutant form of SV40 large T antigen which (a) contains a replication-competent binding site for SV40 origin of replication and (b) is negative for binding to wild-type p53 and to retinoblastoma tumor suppressor gene product due to a mutation in a codon in the p53 binding domain of the large T antigen and a mutation in a codon in the RB binding domain of the large T antigen.

12. The method according to Claim 11, wherein residue 107 of the mutant SV40 large T antigen is lysine and residue 402 is glutamic acid.

13. The method according to Claim 11, wherein the mutant form of SV40 large T-antigen also contains a replication-competent binding site for a BK virus origin of replication.

14. The method according to Claim 1, wherein prior to step (b) is the step: compacting each member of the library with a carrier in the presence of a chaotropic salt to a diameter of less than 30 nm, wherein the carrier comprises a target binding moiety conjugated to a nucleic acid binding moiety, wherein the target binding moiety comprises an antibody or a specific binding fragment thereof which binds to a secretary component of a mammalian polymeric immunoglobulin receptor, where the nucleic acid binding moiety comprises of a polycationic polymer comprising positively charged amino acids.

15. The method according to Claim 14, wherein each member of the library comprises a promoter operably linked to an oligonucleotide encoding one or more gene product encoded in the insert.

16. The method according to Claim 15, wherein the promoter is a viral promoter.

17. The method according to Claim 16, wherein the viral promoter is selected from the group consisting of the SV40 promoter, the MMTV promoter, and the CMV promoter.

18. The method according to Claim 14, wherein the target binding moiety is an antibody.

19. The method according to Claim 18, wherein the antibody is a monoclonal antibody.

20. The method according to Claim 14, wherein the polycationic polymer comprising positively charged amino acids is poly-L-lysine.

21. The method according to Claim 1, wherein prior to step (b) is the step: mixing one or more members of the library with a carrier molecule at a chaotropic salt concentration sufficient for compaction of a complex consisting essentially of a single molecule of one member and a sufficient number of carrier molecules to provide a charge ratio of 1:1, in the form of a condensed sphere, whereby unaggregated complexes are formed, wherein each complex consists essentially of a single molecule of a member and one or more carrier molecules.

22. The method of Claim 21, wherein the chaotropic salt is NaCl.

23. The method of Claim 22, wherein the member and the carrier molecule are each, at the time of mixing, in a solution having a salt concentration of 0.05 to 1.5 M.

24. The method of Claim 21, wherein the carrier molecule is a polycation and the molar ratio of the phosphate groups of the member to the positively charged groups of the polycation is in the range of 4:1 to 1:4.

25. The method of Claim 24, wherein the polycation is added slowly to the members, while vortexing at high speed.

26. The method of Claim 21, in which formation of the complexes is monitored to detect, prevent or correct, the formation of aggregated or relaxed complexes.

27. The method of Claim 26, wherein formation of the complexes is monitored by a method selected from the group consisting of electron microscopy, circular dichroism, and absorbance measurement.

28. The method of Claim 21, further comprising the step of: complexing the unaggregated complexes with lipids.

29. The method of Claim 21, wherein the theoretical minimum diameter is calculated using partial specific volume.

30. The method of Claim 21, wherein the theoretical minimum diameter is calculated using X-ray diffraction density.

31. The method of Claim 21, wherein diameter of the complex is measured using uranyl acetate staining and electron microscopy.

32. The method of Claim 21, wherein the carrier molecule comprises a target cell binding moiety.

33. The method of Claim 21, wherein the carrier molecule comprises a target cell binding moiety covalently linked to a nucleic acid binding moiety.